Microbial update
campylobacter

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In 1886 the German bacteriologist Theodor Escherich observed organisms resembling campylobacter in stool samples from children with diarrhoea, and made drawings of small vibrios seen in the intestinal mucous of infants. In 1913 McFaydean and Stockman isolated such vibrios from foetal tissue of aborted sheep. Right up until 1962 spiral bacteria were being isolated from farm animals suffering a variety of ailments, and these were called 'vibrios'. But in 1963, Vibrio fetus was transferred into a new genus – campylobacter. Following this, many of the species previously classified as vibrio were moved into this new genus, including C. jejuni and C. coli.

In 1957, prior to the assignment of the new genus, King and co-workers successfully isolated 'vibrios' from the blood of humans with diarrhoea, this being the first ever isolation of these organisms from any source.

Since that time, the development of selective growth media in the 1970s permitted more laboratories to test stool specimens for campylobacter and it soon became clear that campylobacter species were common human pathogens, indeed in many countries it is acknowledged that campylobacter causes more reported cases of human illness than any other single organism.

What is it?

Campylobacter are Gram negative narrow, long, rod shaped bacteria. They are spiral shaped, do not produce spores and have a single polar flagellum at each end of the cell.

They are motile and exhibit a corkscrew movement when viewed under the microscope. C. jejuni, C. coli and C. lari appear to cause 90% of reported human illnesses.

These species are often called thermotolerant campylobacter as they grow best at 37°-42°C. They cannot grow below 30°C, therefore whilst they can be carried on chilled foods, they are not able to grow on them.

It has been noted that older cultures of the organism become round in shape (the coccoid form), and these have been linked with a viable non-culturable state (the organism is still alive but cannot be cultured on laboratory media).

Campylobacters are unusual as they do need oxygen to grow, but they cannot tolerate the level of oxygen in air. Microbiologists refer to then as microaerophilic.

What does it cause?

Those that consume viable campylobacter in foods may suffer from campylobacteriosis. It has been estimated that the consumption of as few as 500 cells of the organism may be sufficient to cause illness.

Once eaten, the incubation period is typically two to five days, but onset may occur in as few as two days or as long as 10 days after eating contaminated food. The illness usually lasts no more than one week but severe cases may persist for up to three weeks, and about 25% of individuals experience reoccurrence of symptoms.

It is interesting that even though the immediate illness may be resolved in days, the organism may continue to be shed in faeces for up to 12 weeks. The most consistent symptom of campylobacter infection is diarrhoea which may contain blood. Other symptoms include fever, nausea, vomiting, abdominal pain, headache, and muscle pain.

The majority of cases are mild, do not require hospitalisation, and are self-limited. However, Campylobacter jejuni infection can be severe and life threatening, infecting the blood and other organs.

As well as a simple gastrointestinal food poisoning, a number of long term complications (known as chronic sequelae) can sometimes result from a campylobacter infection. Some people can develop a rare disease that affects the body's nervous system called Guillain-Barre Syndrome (GBS).

This can begin several weeks after the diarrhoeal illness, may last for weeks to months, and often requires intensive care. Full recovery is common but some affected individuals may be left with mild to severe neurological damage. It is thought that 44% of cases of GBS are preceded by infection with campylobacter. Another chronic condition that may be associated with campylobacter infection is a form of reactive arthritis.

Where does it come from?

A wide variety of healthy domestic and wild animals can carry campylobacter. The bacteria usually live in the intestines as part of the animal's normal flora, and is shed in the faeces. With a few exceptions, campylobacter species do not cause any signs of illness in the animal host. It is also possible for the organism to survive in raw water sources and food items such as raw milk.

Because campylobacter has so many reservoirs in the environment, food products (especially poultry, beef, and pork) are at risk of contamination.

Raw milk surveys have shown that campylobacter can occur but is easily inactivated by pasteurisation. Produce may also become contaminated with campylobacter if exposed to contaminated water supplies or animal faeces during growth in the field, but such produce can also be contaminated in the kitchen environment, if it comes into contact with contaminated raw meat or meat juices.

Recent survey work within the UK has indicated that around 65% of all poultry purchased in retail stores will be contaminated with campylobacter.

This forms a major challenge to those using poultry, as large quantities of the organism will be carried into food preparation areas, potentially resulting in risks of cross contamination within kitchen environments.

Other sources of the organism within foods have been noted before in this article, but one interesting one is 'bird pecked milk'. Cases of campylobacteriosis have in the past been linked to doorstep milk deliveries, in which small wild birds have pecked through foil caps of milk bottles to gain access to the milk inside. It is known that birds carry campylobacter and it is assumed that they can pass it into the milk, and when eventually drunk, it can cause human campylobacteriosis.

Pet dogs and cats may also carry campylobacter and shed the bacteria in their faeces; the coats of such animals can then become contaminated and thus transfer organisms to human hands.

Campylobacter originates from the gastrointestinal tract of a variety of animals. From this source it can contaminate carcases, water, produce, milk, food preparation areas and human hands. Care when handling and preparing foods is essential to maintain control over the organism and reduce risks of infection.

Controls for campylobacter

Much intensive work has been done by governments and food producers to try to reduce the levels of campylobacter in poultry. This has had varied effects in different countries, but in the UK we still have, according to recent surveys, around 65% of retail raw poultry containing the organism.

Reducing this level in poultry flocks will not be easy and could take some time, but poultry producers and food retailers are undertaking intensive research in order to achieve this. However, this does not mean we have no effective controls. Industrially cooked poultry production successfully reduces the risk of campylobacter in cooked products, by use of strict hygiene standards, proper cooking and separation of raw from cooked meat.

This regime is very successful and we rarely, if ever, see issues of industrially produced cooked poultry being linked to any cases of food poisoning. In smaller kitchens, careful control of raw poultry (and its packaging material) by ensuring that it (or its juices) do not contact ready to eat foods (RTE), food preparation areas or utensils used for RTE foods are essential. Proper cooking of raw poultry and poultry products (to centre temperatures of 70°C for two minutes) will eliminate viable organisms.

Cooked poultry must then be stored in a way that prevents recontamination. The successful control of the organism in the kitchen involves no special requirements, just thought and good hygienic practices.

Methods of detection

Methods to detect campylobacter have been developed over many years and are detailed in various British Standards, European Standards, International Standards and in the USA, FDA and USDA procedures. Most of these methods are presence or absence tests (the results will say if the organism is present in a known weight of product), are based around the use of conventional broths and agars and can give a result in around 3-5 days.

These methods usually require an enrichment and an isolation on one or more selective agars. This will result in either a negative result, or a presumptive positive.

More recently there has been considerable interest in quantifying the number of campylobacter with samples, and this has led to the development of direct plating methods which allow numbers to be counted. Again these enumeration tests give a presumptive result that has to be confirmed. This is achieved using biochemical tests, which can also be used to identify the species of campylobacter involved.

Over recent years a number of more rapid methods have been produced by commercial method producers, and these can now give results in 30-50 hours.

Popular and well validated rapid methods include those based on immunoassay procedures and the polymerase chain reaction (PCR) technique. It is recommended that any company wishing to use a rapid method for campylobacter detection should only consider those that are well validated (for example by MicroVal, AFNOR, NordVal or AOAC) and they should also check that

these methods work with their own ingredient/product types.

Conclusions

Although campylobacter were first observed in the late 19th Century, they were only cultured in the late 1950s and their role in foodborne illness only became apparent in the 1970s. But we now see them as the cause of more cases of foodborne illness than any other organism. Interestingly, few outbreaks of illness appear to be caused by campylobacter.

The primary source of the organism is the animal gut and, in foods, contaminated raw poultry appears to be a major risk factor.

However, as long as we understand the risk, it is possible to put in place adequate controls. Preventing contact between raw poultry (and its packaging) and RTE products, food preparation areas used for RTE foods, and utensils is key.

The correct cooking of raw poultry and poultry products, and then storage to prevent post cooking contamination, is essential, as is good personal hygiene.

All of these will considerably reduce the risk of campylobacteriosis. ■

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